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Hypothalamic-Pituitary-Adrenal Reactivity to Acute Stress: an Investigation into the Roles of Perceived Stress and Family Resources

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Abstract Rurally situated African Americans suffer from chronic exposure to stress that may have a deleterious effect on health outcomes. Unfortunately, research on potential mechanisms that underlie health disparities affecting the African American community has received limited focus in the scientific literature. This study investigated the relationship between perceived stress, family resources, and cortisol reactivity to acute stress. A rural sample of African American emerging adults ($N = 60$) completed a battery of assessments, the Trier Social Stress Test (TSST), and provided four samples of salivary cortisol: prior to receiving TSST instructions, prior to conducting the speech task, immediately following the TSST, and 15–20 min following the TSST. As predicted, cortisol levels increased in response to a controlled laboratory inducement of acute stress. Moreover, diminished levels of family resources were associated with blunted cortisol reactivity to acute stress. Of note, higher levels of perceived stress over the past month and being male were independently associated with lower levels of cortisol at baseline. Lack of family resources had a blunting relationship on the hypothalamic-pituitary-adrenal axis reactivity. These findings provide biomarker support for the relationship between family resources—an indicator associated with social determinants of health—and stress physiology within a controlled laboratory experiment. Identifying mechanisms that work toward explanation of within-group differences in African American health disparities is both needed and informative for culturally informed prevention and intervention efforts.

Keywords African Americans · HPA axis · Cortisol · Stress · Health disparities

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Introduction

African Americans suffer disproportionate rates of disease-related morbidity and mortality. When compared to European Americans, African Americans have a 30% higher mortality for heart disease, 25% higher mortality for cancer, and 41% higher mortality for stroke. African Americans are also eight times more likely to be diagnosed with HIV and at 60% higher risk of developing diabetes (CDC 2011). The economic burden associated with these health disparities amounts to more than US$100 billion annually (LaVeist et al. 2011). Among rural African Americans, stressors such as experiences of discrimination, lack of access to care, marginalization by health care providers, unemployment, and financial strain magnify health risks and further contribute to disease progression (Appel et al. 2005; Downey 2013; Hartley 2004). Indeed, recent studies have found health disparities in rural communities that are surpassing what is taking place in urban settings (Cossman et al. 2010; Singh and Siahpush 2014). Given this alarming public health problem, a growing body of literature has implicated stress-related allostatic load mechanisms in disease vulnerability (see Juster et al. 2010, for review). The present study aims to characterize how two social determinants of health—perceived stress and low family

resources—impact stress reactivity among rural African American emerging adults.

Physiology of Stress

Allostatic load refers to wear and tear on the body's stress response system (McEwen 1998) in response to chronic, extreme, or incessant real or perceived threats in the environment, particularly unpredictable and social threats. Physiological activation is adaptive in that it helps the body mobilize a near immediate fight-or-flight response and acute long-lasting (over the course of minutes to hours) adjustment of the hypothalamic-pituitary-adrenal (HPA) axis (Sapolsky et al. 2000). This comprises allostasis, the body's capacity to effectively recover from stressful experiences (McEwen and Wingfield 2003), and the HPA axis is particularly well suited to helping return physiological systems to baseline through counter-regulatory processes (e.g., negative feedback; Koob and Le Moal 2008). Over prolonged periods of time, incessant exposure to stressors can alter the HPA axis as the individual attempts to achieve allostasis despite the onslaught of stressors and social challenges in their environment. HPA axis dysregulation can be marked by various patterns of biomarker release, including heightened and blunted reactivity, and may be influenced by adaptive capacities inherent to the individual's experience (Del Giudice et al. 2011; Heim and Nemeroff 1999). Importantly, putative dysregulation is not physiological maladaptation but rather adaptation of the individual's physiology to a difficult or undesirable social environment (Ellis et al. 2013). Adaptive does not mean desirable but rather shifts the focus on the social determinants of health in order to show how the dysregulation of an individual's physiological response unfolds in response to stressful environments (Shirtcliff et al. 2014).

Cortisol, the primary hormonal end product of the HPA axis released from the adrenal gland, is thought of as a stress biomarker. Cortisol release is beneficial as it has been associated with enhancing functioning of the cardiovascular and anti-inflammatory aspect of immune systems, functions to change glucose metabolism, and impacts affective and cognitive function to help the body cope with stress. Because cortisol reaches several biological systems, dysregulations in its output leave the person at risk for many stress-related diseases (Lupien et al. 2009). African Americans may be at increased risk for HPA dysregulation and subsequent disease vulnerability due to a disproportionate exposure to chronic stressors experienced through racism, discrimination, violence, crime, neighborhood disorganization, unemployment, access to care, financial strain, and low socioeconomic status (Clark et al. 1999; Williams et al. 2010).

Despite clear disparities in health and stress exposure among rural African Americans, the stress physiology of this population remains understudied. Previous research has identified dysregulations in basal and diurnal cortisol as well as cortisol awakening response (CAR) among African Americans (Bennett et al. 2004; Cohen et al. 2006; DeSantis et al. 2007; Martin et al. 2012; Obasi et al. 2015; Skinner et al. 2011), but cortisol reactivity to acute stress in this population remains largely understudied (Hostinar et al. 2014a). Dysregulations in acute stress reactivity occur secondary to allostatic states by demonstrating an inability to elicit short-term biological changes in order to meet the demands of the proximal social context. The present study therefore aims to characterize acute stress reactivity among rural African American emerging adults. Because activation of stress responses is ultimately rooted in individual appraisals of threat (Lupien et al. 2009; McEwen 1998), this study will also investigate the relationship between social determinants of health and acute stress reactivity, in order to elucidate individual differences in risk and resilience among this group. Specifically, this study will focus on the unique and collective contributions of perceived stress and family resources.

Perceived Stress and Acute Stress Reactivity

According to allostatic load models, physiological stress activation occurs secondary to the brain's interpretation or appraisal of an environmental stressor (Juster et al. 2010; Lupien et al. 2009). Subjective perception of stress is therefore equally, if not more, important than objective indicators of stress in enacting physiological response. Consistent with such models, Clark et al. (1999) found that perceived stress, above objective environmental stress, was positively correlated with higher allostatic load primary mediators. Goldman and colleagues (2005) further demonstrated that both current and longitudinal perceived stress were positively associated with an index of allostatic load (a measure of cumulative physiological dysregulation) among older adults. Moderate associations between reported perceived stress and altered diurnal cortisol levels have also been consistently evidenced in diverse samples (e.g., Bennett et al. 2004; O'Brien et al. 2013). Despite these findings, the impact of ongoing perceived stress on physiological reactivity to acute stress is understudied among African American emerging adults, whose vulnerability to chronic stress exposure is further compounded by age-related transitional stressors. Identifying changes in acute stress reactivity as a function of perceived stress may help indicate subtle, yet meaningful, allostatic dysregulations that are only detectable when the system is challenged (Kudielka and Wüst 2009).

Impact of Family Resources on Stress

The availability of resources such as food, shelter, financial stability, positive relationships, and quality time with family are integral components to healthy development. Growing up

in environments low on these resources can activate prolonged stress responses that have negative ramifications for physical and mental health through adulthood (Bradley and Corwin 2002; Hostinar and Gunnar 2013) through two main theorized mechanisms. First, diminished family resources can act as a source of stress. For instance, self-reported low social class is linked to lower cortisol reactivity to acute experimental stressors (Kristenson et al. 2001). Low SES is also associated with lower basal cortisol, flatter diurnal rhythms, and higher CAR among adults, suggesting that the effects of low family resources carry over into adulthood and evidence themselves in daily regulatory mechanisms of cortisol output (Cohen et al. 2006; Gustafsson et al. 2011; Steptoe et al. 2003). Individuals with high familial adversity show diminished cortisol and heart rate responses to psychosocial stress, even when diurnal cortisol curves are normal (Lovallo et al. 2012). Second, family-level social support can also affect stress physiology such that social support can attenuate HPA axis reactivity to acute stress, particularly among females (Heinrichs et al. 2003; Kirschbaum et al. 1992). The presence of family resources may help provide the individual with the internal and external resources needed to cope with stressors, allowing their family to buffer them from a physiological stress response. Interestingly, Uhart and colleagues (2006) found heightened cortisol reactivity in European American, but not in African American, participants who grew up in a stressful family context. A recent study has found waking cortisol levels buffered by positive parenting primarily within European American emerging adults more so than African American youth (Shirtcliff et al. in press). This finding suggests important ethnic group differences that further highlight the need to assess acute stress physiology among African Americans.

The Present Study

In context of these variable findings and noted gaps in the literature, this study sought to characterize the trajectory of acute stress reactivity among a vulnerable and understudied population, rural African American emerging adults. The present study also aims to identify psychosocial contributors that are associated with physiological reactivity to acute stress exposure, namely perceived stress and family resources. Based on previous research findings, the following hypotheses were made: (1) Rural African American emerging adults will exhibit blunted cortisol levels when experiencing higher levels of perceived stress; and (2) HPA axis reactivity to acute stress will be lower among participants with low family resources. When designing this study, an a priori power analysis was conducted based on a meta-analysis (Dickerson and Kemeny 2004) that found fewer than 50 participants were needed to obtain 80% power in obtaining statistical significance in cortisol reactivity to acute stress ($\alpha = 0.05$) from the Trier Social Stress Test (TSST). Of note, we utilized a conservative approach to hierarchical linear model (HLM) statistical power by basing the sample size on the level 2 units of analyses (i.e., number of participants) as opposed to level 1 (i.e., total number of saliva samples collected).

Methods

Participants

Participants ($N = 60$) consisted of rural African Americans between the ages of 18 and 22 ($X = 20.0$, $SD = 1.1$). The majority of the participants were female ($n = 39$, 63.9%), unmarried ($n = 58$, 95.1%), and self-identified as fifth generation in response to immigrant status ($n = 54$, 88.5%). In response to highest level of education obtained, 1.7% had some high school education, 30.0% graduated from high school, 55.0% had some college or technical classes, 11.7% had a college degree, and 1.7% had some professional training. Moreover, 62.3% of the participants were currently a homemaker, student, or unemployed. Each participant was asked: "Have you ever used professional services that were provided by a psychologist?" Ninety-five percent ($n = 57$) reported no, and 5.0% ($n = 3$) reported yes.

Procedures

Participants were enrolled into this study after previously participating in a control group from the Adults in the Making (AIM) project. This group represents a random sample of African Americans who reside in rural counties throughout the state of Georgia. Participants who agreed to participate in this study were greeted by a graduate research assistant when they arrived at the laboratory at 2:45 P.M. After obtaining informed consent, participants were asked to (1) provide a breath alcohol sample and urine sample to test for the presence of alcohol, THC, cocaine, opioids, methamphetamine, benzodiazepines, barbiturates, and oxytocin. Participants who tested positive for a licit or illicit substance were not permitted to continue in this session ($n = 2$) and were not included in the participant data presented above. Following a negative drug screen, the participants were asked to complete a battery of assessments, TSST, and provide saliva samples. Finally, the participants were debriefed and financially compensated US$175 for their participation in this study and to offset travel expenses. This study was approved by the university's Institutional Review Board.

Trier Social Stress Test The TSST was used to induce the participants into a state of acute stress. Studies have repeatedly demonstrated that cortisol shows reactivity to the TSST (De Wit et al. 2003; Uhart et al. 2006). During the speech

component, participants were instructed to introduce themselves to the "committee" in a free speech (5 min) and convince them why they should be hired for a job vacancy during these tough economic times. This committee was introduced as being experts in nonverbal behavior and consisted of an African American and European American confederate. The participants were given 5 min to prepare their speech. Once the speech task began, the committee asked standardized questions if the participants paused for more than 20 s prior to the expiration of the speech period. Next, during the arithmetic component, participants were asked to serially subtract the number 13 from 1,022 as quickly and as accurately as possible (5 min). On every failure, participants were prompted to restart at 1,022 with a committee member interfering, "Stop. 1,022."

Salivary Cortisol Four samples of salivary cortisol were collected during the TSST: Sample no. 1 was collected prior to receiving the TSST instructions after arrival at the laboratory and completing some computerized assessments, sample no. 2 was collected prior to conducting the speech task, sample no. 3 was collected immediately following the TSST, and sample no. 4 was collected 15–20 min following sample no. 3. Peak cortisol levels are found in saliva 20–30 min after the experience of an acute stressor, so sample no. 4 was designed to represent the peak cortisol response (PCR) to the TSST.

Measures

Salivary Cortisol All saliva samples were stored immediately in an ultracold laboratory freezer (−30 °C) then shipped overnight frozen with dry ice pellets to the Middleton Research Biodiagnostics Lab (Madison, WI). On the day of assay, samples were thawed and cortisol was assayed in duplicate using a well-established enzyme-linked immunosorbent assay (ELISA) kit specifically designed for use with saliva (Salimetrics, State College, PA). Samples were reanalyzed if the CV for the duplicate measurements were >20%. Samples from the same individual were all assayed on the same run. To normalize distributions, extreme values of raw cortisol were winsorized.

Perceived Stress Stress was measured by the Perceived Stress Scale (PSS; Cohen et al. 1983). The PSS is a 14-item self-report measure that assesses the individuals' perception of situations in their lives that they deem stressful over the past month. The PSS is rated on a five-point Likert scale ranging from "never" to "very often." Summary scores for the PSS range between 0 and 56: with higher scores indicating more stress. Each item on the PSS assesses one's perceived stress within the last month. In previous research, scores on the PSS demonstrated adequate internal consistency and test-retest reliability (Reis et al. 2010). The Cronbach's alpha for scores produced by the PSS was 0.75 in this sample.

Family Resources Family resources was measured by the Family Resource Scale (FRS; Dunst and Leet 1987). The FRS is a 30-item self-report measure that assesses an individual's perceived adequacy of concrete resources in the household. Resources include physical necessities and shelter (eight items), growth and support (nine items), necessities and health (seven items), interfamily support (two items), child care (two items), and personal resources (two items). The FRS is rated on a five-point Likert-type scale ranging from "not at all adequate" to "almost always adequate." A response of "does not apply" was also permitted. Lower scores on the FRS indicate lower perceived adequacy of resources in the childhood home. In previous research, scores on the FRS demonstrated adequate internal consistency, split-half reliability, and criterion-related validity (Brannan et al. 2006; Dunst and Leet 1987). The Cronbach's alpha for scores produced by the family resources scale was 0.95 in this sample.

Analytic Strategy

A two-level HLM was used to estimate peak cortisol reactivity in response to the TSST. Thus, the within-subject dependent variable included all four samples of salivary cortisol from baseline to 15–20 min after the completion of the TSST. To understand how cortisol changed across the session, predictors of cortisol were included at the within-subjects level (i.e., level 1). A random effect for time since baseline (TSB) was modeled in minutes and captured variation in time associated with the collection of saliva samples across the session. A positive TSB indicates a rise in cortisol across the entire session. Cortisol reactivity, however, is not expected to be a gradual rise. Therefore, the PCR to the TSST was modeled using a dummy variable that was coded one for sample no. 4 and zero for the remaining samples. A positive PCR indicates a larger rise after the TSST than expected by the change during the rest of the session. Of note, this is a conservative approach to estimating reactivity in which cortisol may be elevated above baseline and above a general laboratory session-related cortisol rise. Lastly, the intercept in this model (β_0) indexed cortisol levels (sample no. 1) at baseline prior to the start of the TSST.

Individual difference predictors were then modeled as cross-level interactions between these cortisol parameters and variables of interest (i.e., perceived stress or family resources). Perceived stress, family resources, age, and sex were entered as main effects on baseline cortisol levels. Moreover, family resources was entered as a main effect on PCR after controlling for perceived stress, family resources, age, and sex at baseline. None of the predictors was found to influence the variation in time associated with the collection of saliva

samples (β_1) since this took place in a controlled laboratory setting.

Level 1 (within individual)	Cortisol = $\beta_0 + \beta_1$ (TSB) + β_2 (PCR) + r
Level 2 (between individual)	$\beta_0 = \gamma_{00} + \gamma_{01}$ (age) + γ_{02} (sex) + γ_{03} (PSS) + γ_{04} (FRS) + U_0
	β_1 (TSB) = $\gamma_{10} + U_1$
	β_2 (PCR) = $\gamma_{20} + \gamma_{21}$ (FRS) + U_2

TSB time since baseline, *PCR* peak cortisol response, *PSS* Perceived Stress Scale, *FRS* Family Resource Scale

Results

Descriptive Statistics

Scores on the PSS ranged from 8 to 38 ($M = 24.62$; $SD = 6.26$), and scores on the FRS ranged from 38 to 163 ($M = 135.81$; $SD = 24.92$). Moreover, the PSS was not significantly correlated to the FRS ($r = -0.16$, $p = .237$). Mean cortisol levels were 0.14 μl/dl($SD = 0.07$) at baseline, 0.14 μl/dl ($SD = 0.07$) prior to starting the TSST, 0.19 μl/dl ($SD = 0.12$) at the end of the TSST, and 0.25 μl/dl ($SD = 0.17$) 15–20 min following the TSST. Of note, peak cortisol reactivity to acute stress represented a large effect size ($d = 1.51$) in this study.

Stress and Cortisol Levels

Trait (systematic) cortisol comprised a significant proportion (39.7%) of the total variability in cortisol reactivity ($\chi^2(57) = 205.47$, $p < .001$). This suggests that rural African American emerging adults have moderately stable salivary cortisol levels in response to acute stress. TSB represented a significant linear slope from the baseline sample to the peak sample ($\beta = 0.002$, $t(57) = 4.80$, $p < .001$). On average, cortisol levels increased in response to the controlled laboratory stressor via the TSST. Furthermore, 29.3% of the total variance in cortisol was found to be a function of stable systematic individual differences after accounting for time in the model ($\chi^2(57) = 155.76$, $p < .001$). PCR also represented a significant linear slope to the model ($\beta = 0.02$, $t(57) = 2.36$, $p = .022$). This positive relationship indicated that cortisol levels significantly increased in sample no. 4 which represented the peak cortisol reactivity to the TSST. Moreover, 65.4% of the total variance in cortisol was found to be a function of stable systematic individual differences after accounting for time and the peak cortisol response to the TSST in the model ($\chi^2(56) = 185.11$, $p < .001$).

We tested if some of the variability in cortisol at baseline could be accounted for by age, sex, perceived stress, and family resources. Sex was associated with baseline levels of cortisol ($\gamma_{02} = 0.043$, $t(53) = 2.20$, $p = .032$). Indeed, males were found to have lower levels of cortisol at baseline. Additionally, perceived stress had a significant inverse relationship with baseline levels of cortisol ($\gamma_{03} = -0.003$, $t(53) = -2.14$, $p = .037$). More specifically, experiencing higher levels of perceived stress over the past month was associated with lower levels of cortisol at baseline. Age and family resources were not associated with levels of cortisol at baseline (see Table 1). Of note, post hoc analyses found that there were no sex differences in perceived stress or family resources.

Family Resources and Peak Cortisol Reactivity to Acute Stress

We tested whether some of the variability in PCR could be accounted for by family resources after controlling for age, sex, perceived stress, and family resources at baseline. Family resources had a significant positive relationship with PCR ($\gamma_{21} = 0.001$, $t(56) = 2.30$, $p = .025$). More specifically, coming from a family with diminished levels of basic resources was associated with a blunted peak cortisol response to acute stress (see Fig. 1). Of note, a post hoc analysis found

Table 1 Hierarchical linear models of the relationship between perceived stress, family resources, and cortisol reactivity to acute stress

Model	Fixed effect	Coefficient	*SE*	*t*	*df*	*p* value
1	γ_{00} *(baseline)*	0.175	0.011	15.275	57	*<0.001*
2	γ_{00} *(baseline)*	0.128	0.009	13.973	57	*<0.001*
	γ_{10} *(TSB)*	0.002	4.03E−04	4.803	57	*<0.001*
3	γ_{00} *(baseline)*	0.133	0.010	14.012	57	*<0.001*
	γ_{10} *(TSB)*	0.001	4.16E−04	3.067	57	*0.003*
	γ_{20} *(PCR)*	0.045	0.019	2.357	57	*0.022*
4	γ_{00} *(baseline)*	0.304	0.130	2.330	53	*0.024*
	γ_{01} (age)	−0.009	0.006	−1.465	53	0.149
	γ_{02} *(sex)*	0.043	0.020	2.199	53	*0.032*
	γ_{03} *(PSS)*	−0.003	0.001	−2.143	53	*0.037*
	γ_{04} *(FRS)*	−4.65E−04	3.01E−04	−1.546	53	0.128
	γ_{10} *(TSB)*	0.001	4.15E−04	3.076	57	*0.003*
	γ_{20} *(PCR)*	0.045	0.019	2.395	57	*0.02*
5	γ_{00} *(baseline)*	0.304	0.130	2.333	53	*0.024*
	γ_{01} (age)	−0.009	0.006	−1.469	53	0.148
	γ_{02} *(sex)*	0.043	0.020	2.203	53	*0.032*
	γ_{03} *(PSS)*	−0.003	0.001	−2.158	53	*0.035*
	γ_{04} (FRS)	−3.59E−04	2.93E−04	−1.225	53	0.226
	γ_{10} *(TSB)*	0.001	4.18E−04	3.084	57	*0.003*
	γ_{20} *(PCR)*	0.045	0.019	2.430	56	*0.018*
	γ_{21} *(FRS)*	0.001	4.61E−04	2.295	56	*0.025*

PSS Perceived Stress Scale, *FRS* Family Resource Scale, *TSB* time since baseline, *PCR* peak cortisol response

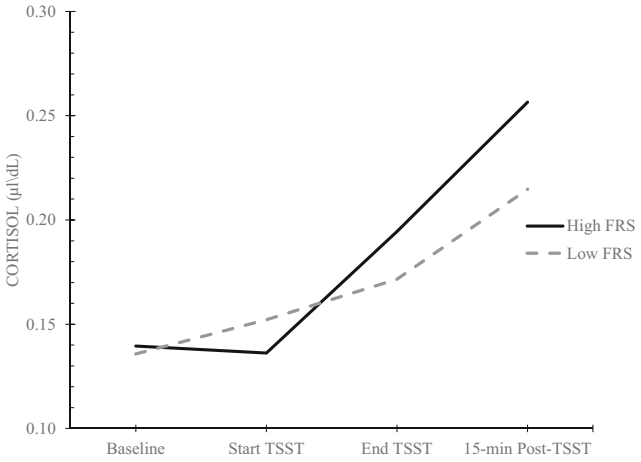

Fig. 1 Relationship between family resources and mean cortisol reactivity to acute stress at each time point during the TSST. *TSST* Trier Social Stress Test, *FRS* Family Resource Scale

that perceived stress was not associated with the peak cortisol response to acute stress.

Discussion

This study sought to extend the existent literature by investigating the relationship between psychosocial determinants of health, specifically family resources and perceived stress, and HPA reactivity to acute stress by utilizing a controlled laboratory experiment. To our knowledge, this is the first study to investigate within-group variation in psychosocial determinants of health that utilized a controlled laboratory assessment of acute stress with a community sample of rural African American emerging adults. The results were consistent with previous research studies that investigated perceived stress as an indicator of allostatic load (Clark et al. 1999; Goldman et al. 2005). More specifically, participants who reported greater levels of perceived stress over the past month had blunted levels of cortisol at baseline. Previous research has also demonstrated a link between blunted diurnal cortisol output and chronic stress among rural African Americans, suggesting perceived stress has deleterious consequences for stress physiology across both acute and chronic paradigms (Obasi et al. 2015). High exposure to such unmitigated stressors may put this population at increased risk for allostatic "wear and tear" that contributes to negative health outcomes over time. To this end, a previous longitudinal study found that lower cortisol reactivity to acute stress was associated with a greater incidence of obesity, poorer self-rated health, poorer lung function, and lower cognitive function 5 years later (de Rooij 2013). It is important to note that sex was also associated with baseline levels of cortisol. More specifically, African American males tended to have a more blunted cortisol level at baseline in comparison to their female counterparts. Unfortunately, it is difficult to explain this finding given the fact that there were no sex differences in perceived stress or family resources.

This study extends the current literature by also investigating the relationship between social determinants of health (i.e., family resources) and HPA functioning. Participants who reported lower consistency of having access to adequate family resources in the household had a blunted peak cortisol reactivity to acute stress. This is consistent with previous research demonstrating blunted cortisol reactivity to acute stress in individuals with childhood chronic stress exposure (Andreotti et al. 2015). Higher rates of reported exposure to psychosocial stressors within this population undergird the need to investigate causal mechanisms underlying the relationship between psychosocial determinants of health and negative health outcomes. While we emphasize the role of the social environment in blunting physiological responses to acute stress, previous research has also found that a higher perceived sense of social control was positively related to more robust cortisol reactivity in acute laboratory stressors (Barrington et al. 2014). Although beyond the scope of this study, such findings collectively suggest that one's sense of personal control over their social environment, in addition to their social environment itself, may be a key component in either exacerbating or mitigating against the effects of stress.

Theoretical models are increasingly taking a dynamic and functional view of stress biomarkers that allow for high (or low) physiological arousal to have both costs and benefits (Hostinar and Gunnar 2013). High cortisol appears adaptive when being open to the environment that allows for greater encoding of positive social information (Del Giudice et al. 2013). For example, pharmacological administration of cortisol or a rise in cortisol after awakening is associated with energy, productivity, and reduced symptoms of fatigue (Adam et al. 2006; Chida and Steptoe 2009; Lovas and Husebye 2003). Within acute stressors like the TSST, social memory for salient cues is enhanced within stress-reactive individuals (Wiemers et al. 2013) and pharmacologically blocking a cortisol response is linked with greater self-reports of distress (Andrews et al. 2012). The costs of an "open" or sensitive profile are expected to be minimal in low stress environments. Physiological attunement, including the HPA axis, allows individuals to benefit from stress buffering provided by a supportive individual (Gunnar and Hostinar 2015; Heinrichs et al. 2003) or psychosocial resources bolstered by a supportive environment (Taylor et al. 2008, 2010).

In more challenging environments, however, the costs outweigh benefits of social sensitivity, and it is adaptive instead to be internally buffered from the onslaught of daily hassles and difficulties. For African Americans, this "buffered" profile may be common given, at minimum, experiences with racism and discrimination (DeSantis et al. 2007; Skinner et al. 2011).

Within the present study, this buffered profile was further apparent within African Americans with greater stress exposure and fewer family resources. A buffered profile has benefits in that social threat and uncontrollability exert a diminished impact on the individual, yet there are also potential costs—many of which take years to accumulate a wear and tear on the individual (Koob and Le Moal 2008). For example, the anti-inflammatory and glucose metabolism functions of cortisol may leave the low-cortisol individual at heightened risk for immune and insulin-related health problems (Miller et al. 2002; Raison and Miller 2003). Importantly, this calibration of the body's stress response system is in response to the individual's social environment and stressor exposure (Voellmin et al. 2015), calling attention to the importance of social determinants of health particularly within the African American community.

Taken together, findings from the present study suggest that ongoing perceived stress, in addition to diminished family resources, can profoundly impact stress physiology. Having greater insight into these causal mechanisms of stress dysregulation is critically important given the growing body of evidence linking stress dysregulation to drug use vulnerability and other poor health outcomes. Stress management is a psychological phenomenon that has been long studied in the social and behavioral sciences. While the elimination of social inequities is likely the most robust strategy for eradicating known health disparities in the African American community, creating and disseminating culturally informed stress management and family-strengthening prevention and intervention efforts could also help mitigate this public health problem.

While this study makes a significant contribution to the health disparities literature, it is not without limitations. First, this representative community sample of rural African Americans was small and recruited from the southeast USA. Extending these finding to other rural—and potentially urban—communities should be done with caution. Additionally, while this study was funded to collect data on peak cortisol reactivity to acute stress, we were unable to collect and assay subsequent cortisol samples that would provide information regarding how efficiently cortisol was downregulated after exposure to a controlled laboratory stressor. Future research should be conducted to investigate these relationships in other geographical regions while also collecting additional cortisol samples to effectively model both the upregulation and downregulation of cortisol reactivity to acute stress in this population. Future research may also investigate the downstream effects of such dysregulation in cortisol reactivity to acute stress as a function of sex. Cortisol, and the HPA axis more broadly, can affect multiple biological systems and is recognized to have meaningful cross talk with other neuroendocrine mechanisms (Marceau et al. 2014). For example, our focus on family resources dovetails well with emerging literature on oxytocin as a protective biomarker (Hostinar et al. 2014b) which is altered within individuals with life stress exposure (Seltzer et al. 2014). However, the impact of sex and such dysregulation on other regulatory functions in the acute phase is not well understood.

Despite some limitations, this study reinforces the need for future research to identify between-group and within-group variation in how psychosocial determinants of health can get "under the skin" and develop different health profiles across time. Identifying explanatory mechanisms linked to health disparities will better position researchers to create culturally informed prevention and intervention strategies aimed at reducing—and ultimately eliminating—health disparities in the African American community.

Compliance with Ethical Standards

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Conflicts of Interest The authors declare that they have no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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